The systemic inflammatory response syndrome, organ failure, and mortality after abdominal aortic aneurysm repair

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Background: **Organ failure is a major cause of morbidity and mortality after abdominal aortic aneurysm (AAA) repair. The aim of this study was to determine the relationships between the systemic inflammatory response syndrome (SIRS), organ failure, and mortality after AAA repair and to determine whether the clinical monitoring of SIRS was a useful adjunct to clinical method.**

Methods: **One hundred consecutive patients undergoing open AAA repair were prospectively studied. Patients were divided into three groups: those undergoing elective AAA repair, those with symptomatic but nonruptured AAA, and those with ruptured AAA. The presence of SIRS and organ failure was recorded on a daily basis for each patient until discharge or death.**

Results: **Most patients had SIRS develop during the postoperative period: 89% of the elective group, 92% of the emergency nonruptured (urgent) group, and 100% of the ruptured group. Multiorgan failure occurred in 3.8% of the elective group, 38% of the urgent group, and 64% of the ruptured AAA group. After ruptured AAA repair, the concurrent absence of both SIRS and any organ failure for 48 hours had a sensitivity of 93% and a specificity of 91% as a predictive indicator of subsequent survival to hospital discharge. Patients in whom multiorgan failure developed after ruptured AAA repair had a significantly higher mortality rate (69%) than those who did not (0%; $P = .001$; 95% CI for the difference, 30.2% to 85.8%).**

Conclusion: **The differences in the incidence rate of multiorgan failure between the patient groups compared with the high incidence rate of SIRS in all patient groups supports the two-hit hypothesis of multiorgan failure. The presence of multiorgan failure after ruptured AAA repair is associated with poor outcome. The absence of SIRS and organ failure in these patients is a good predictive indictor of survival. (J Vasc Surg 2003;37:600-6.)**

Organ failure, either single or multiple, is responsible for a large proportion of the morbidity and mortality associated with abdominal aortic aneurysm (AAA) repair.[1] Postoperative management of patients after AAA repair involves intensive monitoring of organ function and aggressive therapeutic intervention if organ dysfunction or failure occurs. Unfortunately, a significant number of patients in whom organ failure develops may not survive,[2] and those who do survive usually have prolonged stays on critical care wards at considerable financial and human cost.

Early detection and treatment of organ dysfunction may prevent progression to single/multiple organ failure and death.[3] However, patients recovering from AAA repair have varying degrees of altered physiologic, hematologic, and biochemical homeostasis, and the identification of organ dysfunction in these patients is not straightforward.

The systemic inflammatory response syndrome (SIRS) is an initial response to injury and reflects activation of inflammatory cascades[4] with production of systemic inflammatory mediators. SIRS should precede the development of organ failure and therefore may be a useful indicator of impending organ dysfunction. The measurement of inflammatory markers associated with SIRS has been proposed as a method of identification of those at risk of multiple organ failure[5]; however, determination of whether a patient has SIRS or not is considerably easier than the measurement of inflammatory mediators. SIRS is defined by the concurrent presence of two or more of four simple clinical criteria (Table I).[3] SIRS can be rapidly assessed at the bedside without the need for any special equipment or expertise other than sound clinical method. The aim of this study was to determine the incidence rate of SIRS after AAA repair and its temporal relationships with organ failure and mortality in these patients to identify whether the routine monitoring of SIRS may be useful in these patients.

PATIENTS AND METHODS

A prospective study was performed of 100 consecutive patients undergoing conventional (open) AAA repair who survived the initial operation. Patients were classified into three groups: those undergoing elective repair (elective), those undergoing emergency surgery for symptomatic but nonruptured aneurysms (urgent), and those with ruptured aneurysms (ruptured). Urgent AAA repairs were those with patients admitted with symptomatic AAA in whom surgery was performed as an emergency without full preoperative work-up because they were perceived to be at risk of imminent rupture or thought to have actually had rupture

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but subsequently were found not to have ruptured at laparotomy. Aneurysm rupture was defined as the presence of free intraperitoneal blood or retroperitoneal hematoma at laparotomy. All patients with ruptured AAA irrespective of the degree of preoperative cardiovascular collapse were included in the same outcome group. Procedures performed via both the transperitoneal and the retroperitoneal approaches were included as were those patients with suprarenal aneurysms. The study was approved by the local ethics committee.

For each patient, the presence of SIRS and organ failure in the previous 24 hours was recorded on a daily basis from the first postoperative day until hospital discharge or death. Failure in the following organ systems (according to the definitions of Knaus et al[6]) was recorded: cardiovascular, respiratory, renal, hematologic, and neurologic (Table II). Any organ failure was defined as one or more organ systems failing during a single 24-hour period, and multiorgan failure was defined as the presence of two or more organ system failures during the same 24-hour period. When any of the criteria defining SIRS or organ failure had not been, or could not be, measured by the surgical or critical care teams, they were assumed to be within normal limits. The assumption of normality for these criteria was to enable the application of SIRS criteria to those clinical data that are routinely recorded without having to make additional clinical or laboratory assessments. Mortality was defined as death before hospital discharge.

Sedated, ventilated patients were assumed to have the same Glasgow Coma Scale score as they had when ventilation was initiated. Patients with chronic organ impairment (with the exception of dialysis-dependant chronic renal failure) were assessed with the same criteria for organ failure as all other patients.

RESULTS

Patient demographics. One-hundred patients were included in the study (80 male; 80%), with a overall median age of 72 years (range, 48 to 85 years). Sixty-two patients underwent elective aneurysm repair (46 male; 74%), with a median age of 72 years (range, 48 to 85 years); 13 patients underwent urgent repair (12 male; 92%), with a median age of 74 years (range, 62 to 82 years); and 25 patients underwent emergency repair of ruptured AAA (22 male; 88%), with a median age of 72 years (range, 59 to 80 years).

Mortality. Overall, 15 patients (15%) died. The mortality rate was 4.8% in the elective group, 7.7% in the urgent group, and 44.0% in the ruptured aneurysm group. For the entire 100 patients in the study, the mortality rate was significantly higher in those patients with any organ failure (23%) compared with those without (0%; $P = .008$, Fisher exact test; 95% CI for the difference, 11.8 to 36.7) and in those in whom multiorgan failure developed (50%) compared with those in whom it did not (3.9%; $P < .0001$; 95% CI for the difference, 26.2 to 64.8; Table III).

Elective AAA repair Sixty-two patients underwent elective AAA repair. SIRS developed in 55 patients (89%) during the postoperative period, failure of one or more organ systems developed in 29 patients (47%), multiorgan failure developed in three patients (4.8%), and three patients (4.8%) died (one from myocardial infarction, one from perforated ischemic colitis, and one from a mesenteric infarction).

Table I. SIRS criteria

Two or more of following:
Temperature >38° C or <36° C
Heart rate >90 bpm
Respiratory rate >20 breaths/min or $PaCO_2$ <4.3 kPa (32.25 mm Hg)
WBC >12 000 cells/mm^3 or <4000 cells/mm^3 or >10% immature (band) forms

WBC, White blood cells.

Of the 29 patients in whom any organ failure developed, 28 also had SIRS develop. Seven patients had SIRS develop before organ failure, 13 concurrently with organ failure, and eight after organ failure. In total, 20 patients (68.9%) had SIRS develop either before or concurrently with any organ failure compared with nine patients (31.0%) in whom SIRS developed either after organ failure or not at all. Although SIRS was relatively common in the early postoperative period, it rapidly decreased over time in these patients, and organ failure also followed a similar pattern (Fig 1).

Urgent AAA repair. Only 13 patients had urgent repair of symptomatic but nonruptured AAA. Twelve patients (92%) had SIRS develop, seven patients (54%) had failure of one or more organ systems develop, five patients (38%) had multiorgan failure develop, and one patient (8%) died of multiorgan failure.

In the urgent group of those patients in whom any organ failure developed (seven patients; 54.0%), six had SIRS develop before or concurrently with organ failure (85.7%) and one after organ failure. Five patients in the urgent group had multiorgan failure develop. One of these patients had SIRS develop before multiorgan failure, and four concurrently with multiorgan failure. No patient had multiorgan failure develop after urgent AAA repair without SIRS either preceding it or occurring at the same time.

Ruptured AAA. Twenty-five patients had ruptured AAA. All patients in this group had SIRS develop, and all patients had one or more organ failures develop. Sixteen patients (64%) had multiorgan failure develop, and 11 patients died (44%).

In these 25 patients, SIRS preceded any organ failure in six and occurred concurrently with any organ failure in 15. Sixteen patients had multiorgan failure develop, 10 of whom had SIRS develop before multiorgan failure, and the remaining six had SIRS develop concurrently with multiorgan failure. SIRS, therefore, occurred concurrently or before organ failure in 84% of those patients in whom organ failure developed and in 100% of patients in whom multiorgan failure developed. In contrast to the elective aneu-

Table II. Definitions of organ failure according to Knaus et al[6]

Organ system	*Criteria for failure*
Cardiovascular	Heart rate ≤ 54 bpm Mean arterial pressure ≤ 49 mm Hg Ventricular tachycardia or ventricular fibrillation pH ≤ 7.24 and $PaCO_2$ ≤ 49 mm Hg (6.57 kPa)
Respiratory	Respiratory rate ≤ 5 breaths/min or ≥ 49 breaths/min $PaCO_2$ ≥ 50 mm Hg (6.58 kPa) A_aDO_2 ≥ 46.5 Dependant on ventilator on 4th organ failure day (ie, not applicable until after 72 h organ failure)
Renal (unless on chronic dialysis before admission)	Urine output < 480 mL/24 h or < 160 mL/8 h Serum urea ≥ 16.6 mmol/L Serum creatinine ≥ 308 μmol/L
Hematologic	Leucocyte count ≤ $1 \times 10^3/mm^3$ Platelet count ≤ $20 \times 10^3/mm^3$ Hematocrit ≤ 20%
Neurologic	Glasgow coma scale ≤ 6 (in absence of any sedation at any one point in day)

One or more positive variables in each category during 24-hour period constitutes that organ failing on that day.
A_aDO_2, Alveolar-arterial oxygen difference.

Table III. Mortality rates for patients with and without SIRS, organ failure, and multiorgan failure values are percentages. Note: all patients with ruptured AAA had SIRS and organ failure develop.

	SIRS				*Organ failure*				*Multiorgan failure*			
	Yes	*No*	*Difference (95% CI)*	P *value**	*Yes*	*No*	*Difference (95% CI)*	P *value**	*Yes*	*No*	*Difference (95% CI)*	P *value**
All patients (n = 100)	16.3	0.0	16.3 (−16.7 to 25.2)	.602	23.0	2.0	21.0 (8.6 to 33.0)	.008	50.0	3.9	46.1 (26.2 to 64.8)	<.0001
Elective (n = 62)	5.5	0.0	5.5 (−30.2 to 14.9)	1.00	6.9	3.0	3.9 (−9.4 to 19.1)	.595	0.0	3.1	-3.1 (−8.7 to 53.1)	1.000
Urgent (n = 13)	8.3	0.0	8.3 (−71.3 to 35.4)	1.00	14.3	0.0	14.3 (−26.5 to 51.3)	1.000	20.0	0.0	20.0 (−16.3 to 62.4)	.385
Ruptured (n = 25)	–	–	–		–	–	–		68.8	0.0	68.8 (30.2 to 85.8)	.001

*Fisher exact test.

rysm group (Fig 1)—although again, SIRS was relatively common in the early postoperative period in the ruptured aneurysm group—it persisted rather than resolved rapidly (Fig 2), as did any organ failure and multiorgan failure.

Multiorgan failure was either the cause of or a significant factor in most deaths after ruptured aneurysm repair (10 of 11 deaths; 91%; the other death from a cerebral infarction). Those patients in whom multiorgan failure developed had a significantly higher mortality rate (69%) than those in whom it did not (0%; $P = .001$; 95% CI for the difference, 30.2 to 85.8; Table III).

The mortality rate in the 20 patients in whom cardiovascular failure developed was 55.0% (11 patients), compared with 0.0% (0 of five patients) in those in whom it did not ($P = .05$, Fisher exact test). In those patients in whom renal failure developed (16 patients), the mortality rate was 62.5% (10 patients), compared with 11.1% (one of nine patients) in those in whom it did not ($P = .03$, Fisher exact test). No significant difference was seen in mortality rate between those patients with and without development of respiratory, hematologic, or neurologic failure ($P = 1.0$, .44, and .44, respectively).

In this group, 12 patients had recurrent multiorgan failure develop after resolution of the initial episode. Of these 12 patients, 10 did not undergo resolution of SIRS before development of recurrent multiorgan failure. Two patients had multiorgan failure develop despite previous SIRS negativity for 24 hours. One of these patients had SIRS develop 2 days before development of recurrent multiorgan failure, but the other had SIRS develop on the same day as multiorgan failure. In this group, the absence of SIRS and the absence of SIRS or any organ failure for 24, 48, and 72 hours in relation to subsequent mortality was examined.

Seven patients with ruptured AAA died despite SIRS having resolved for at least 24 hours. This number decreased after 48 and 72 hours of resolution of SIRS to six and four, respectively. Only two patients died after SIRS and organ failure had resolved for 24 hours, and this decreased to one patient (who died of a cerebrovascular

accident) if the "absence time" was increased to 48 and 72 hours. The resolution of SIRS and the resolution of both SIRS and organ failure were examined as predictive tests for survival after ruptured aneurysm repair. The sensitivity, specificity, positive predictive value, negative predictive value, and likelihood ratio for each test criteria are shown in Table IV. In isolation, the resolution of SIRS is a sensitive test for prediction of survival after ruptured AAA repair but has low specificity. If the resolution of any organ failure is included in the test criteria, the specificity of the test increases substantially, with only a slight reduction in sensitivity.

Variation between patient groups Fig 3 shows the incidence rates of SIRS, any organ failure, and multiorgan failure in the three patient groups (elective, urgent, and ruptured). Although SIRS was common in all patients, its prevalence varied with time in each group (Fig 4). In the elective and urgent groups, the prevalence of SIRS rapidly declined in the early postoperative period and remained low, but in the ruptured aneurysm group, the presence of SIRS persisted in a significant number of patients over a prolonged period.

The median time to the onset of both SIRS and any organ failure was 1 day for all groups of patients. In the elective and ruptured groups, the median time to onset of multiorgan failure was 3 days, and in the urgent group, it was 1 day. The median times to onset of SIRS (1 day), any organ failure (1 day), and multiorgan failure (3 days) were significantly different in the ruptured aneurysm group ($P = .007$, Kruskal-Wallis test).

Cardiovascular failure was the most common organ failure overall, occurring in 50% of patients; respiratory failure occurred in 34%, and renal failure in 24%. Neurologic failure and hematologic failure were relatively uncommon, occurring in 2% and 1% of patients, respectively. The relative incidence rates of cardiovascular, respiratory, and renal failure were different in the three groups of patients (Table V). Cardiovascular and respiratory failure occurred with similar rates in both the ruptured and urgent groups, and in the elective group, cardiovascular failure was three times as common as respiratory failure. Renal failure was far more common in the ruptured group than either the urgent or elective groups.

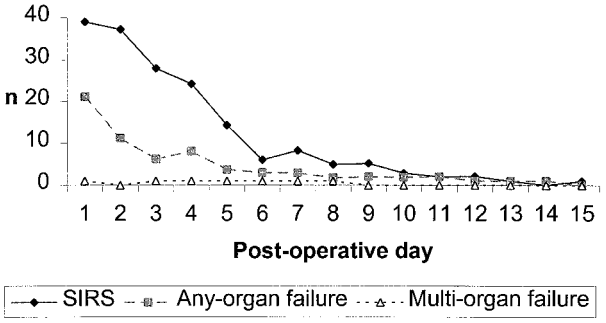

Fig 1. Number of patients with SIRS, any organ failure, and multiorgan failure in elective aneurysm group during first 15 postoperative days.

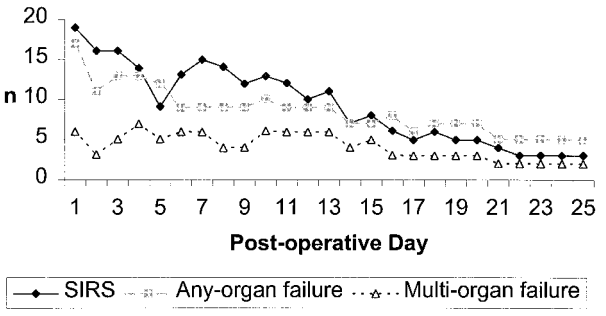

Fig 2. Number of patients with SIRS, any organ failure, and multiorgan failure in ruptured aneurysm group during first 25 postoperative days.

DISCUSSION

This prospective study shows that SIRS and organ failure are common after aneurysm repair, particularly after surgery for rupture. In all patients, the development of multiorgan failure, and in the ruptured AAA group, the development of either cardiovascular or renal failure, were significantly associated with mortality. SIRS occurs concurrently with or before any organ failure in most cases, and also, the resolution of both SIRS and organ failure at any point during the postoperative period is a useful prognosticator of a successful outcome after ruptured aneurysm repair.

The difference in the incidence rates of any organ failure and multiorgan failure between the groups of patients is marked, but the incidence rate of SIRS in each group is similar. Because, by definition, SIRS should precede the development of organ failure, it may be expected that most patients would have organ failure develop, but this is not the case, and a similar finding has been shown in patients in the general intensive care unit.[7] The most obvious clinical difference between these groups is the nature of the insults that each receives. All patients are subjected to the surgical trauma of an AAA repair. In the urgent group, the patients are unselected and frequently not afforded the same degree of preoperative preparation as the elective group. The ruptured group also has a varying degree of hemorrhagic shock before surgery. Given the marked differences between the ruptured group and all other patients, it would appear to be the dual insult of hemorrhagic shock together with surgery that is responsible for the higher incidence rate of organ failure, a theory that is supported by experimental studies.[8]

One of the limitations of this study is the relatively small number of patients in the ruptured AAA group. Unfortunately, this group was the only group with a significant incidence rate of multiorgan failure. It would have been desirable to examine the temporal changes in the degree and type of organ failure in those in whom multiorgan

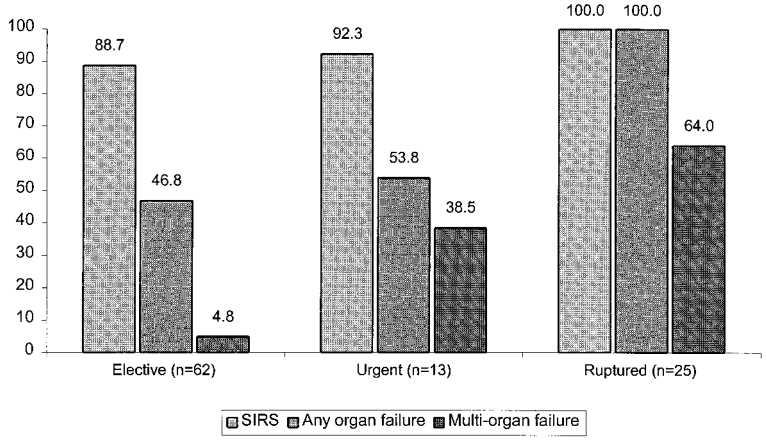

Fig 3. Incidence rates of SIRS, any organ failure, and multiorgan failure in elective, urgent, and ruptured aneurysm groups.

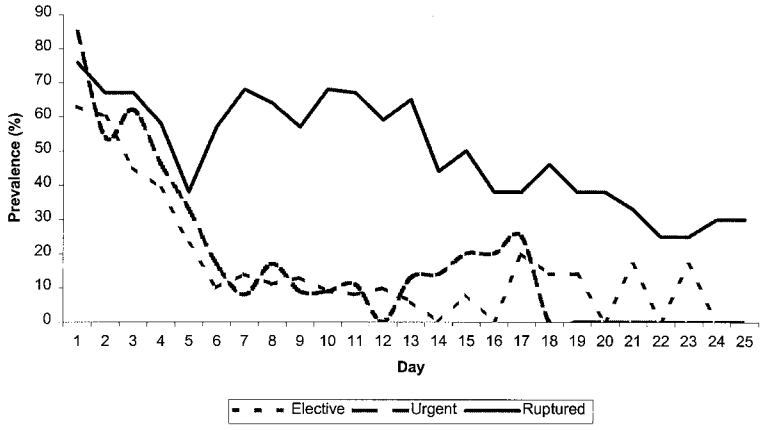

Fig 4. Incidence rate of SIRS by patient category over time (first 25 days after surgery).

Table IV. Resolution of SIRS compared with resolution of SIRS and organ failure for three time periods as predictive tests of survival after ruptured AAA repair

	SIRS resolved for			*SIRS and organ failure resolved for*		
Test criteria	*24 h*	*48 h*	*72 h*	*24 h*	*48 h*	*72 h*
Sensitivity (95% CI)	100% (74.1 to 100)	100% (74.1 to 100)	100% (74.1 to 100)	81.8% (52.3 to 94.9)	90.9% (62.3 to 98.4)	90.9% (62.3 to 98.4)
Specificity (95% CI)	35.7% (16.3 to 61.2)	42.9% (21.4 to 67.4)	64.3% (38.8 to 83.7)	71.4% (45.4 to 88.3)	92.9% (68.5 to 98.7)	92.9% (68.5 to 98.7)
PPV (95% CI)	0.55 (0.34 to 0.74)	0.58 (0.36 to 0.77)	0.69 (0.44 to 0.86)	0.69 (0.42 to 0.87)	0.91 (0.62 to 0.98)	0.91 (0.62 to 0.98)
NPV (95% CI)	1.0 (0.57 to 1.0)	1.0 (0.61 to 1.0)	1.0 (0.70 to 1.0)	0.83 (0.55 to 0.95)	0.93 (0.69 to 0.99)	0.93 (0.69 to 0.99)

PPV, Positive predictive value; *NPV,* negative predictive value.

failure developed, but because of the low numbers of patients in this group, inferences from any analysis performed on this subgroup would be flawed. Therefore, these results have not been analyzed in this manner.

Theories regarding the pathogenesis of organ failure in response to inflammatory insults concentrate around the host response to the initial insults and the magnitude, number, and timing of these insults.[9] An inflammatory

response is essential for the continued survival of a patient. Organ dysfunction and failure in response to injury are, in part, the result of the excessive activation of inflammatory pathways, and antiinflammatory mechanisms exist to minimize this inflammation. Bone[10] describes this as the concept of a compensatory antiinflammatory response syndrome (CARS). If SIRS and CARS are in balance, the inflammatory process is stopped, but if SIRS predominates, proinflammatory pathways become excessively activated. The host response (CARS) is largely constant, but the initial proinflammatory response may vary. If an initial insult causing SIRS is limited by CARS, homeostasis is maintained. However, this initial insult may cause priming of inflammatory pathways, such that a second later insult causes a degree of inflammation greater than that which would be expected if the second insult were to occur in isolation. This is referred to as the two-hit phenomenon. This second hit is said to occur some days after the initial insult, and if this were the case, this theory would fail to explain the differences seen between the groups in this study. However, more recently, the timing required of a second hit to cause excessive inflammation has been shown not to be limited to late in the inflammatory history but also to occur in the first few hours,[11] which is supported by the data presented previously—the initial insult being the hemorrhage associated with aneurysm rupture and the second being the ischemia-reperfusion injury as a result of aortic aneurysm repair.

At first,[4] SIRS was defined as part of a continuing inflammatory response that could have progressed from SIRS to sepsis if microbiologically proven infection occurred and on to severe sepsis if systemic hypoperfusion occurred. Although these subclassifications may have had a better predictive ability than SIRS alone, the subjectivity in their definition adds complexity to SIRS, which alone is an objective and simply defined phenomenon.

A potential confounding factor in interpretation of the results of this study is the effects of the operation itself. SIRS cannot be considered pathognomonic for inflammation. A normal physiologic response to hypovolemia is tachycardia and tachypnea, the presence of which alone would categorize a patient as having SIRS, and in addition, patients undergoing AAA repair are often hypothermic at the end of the procedure, a further positive SIRS criteria. The high incidence rate of SIRS in the immediate postoperative period may reflect this, and when originally defined, the qualification that SIRS "should represent an acute alteration from baseline in the absence of other known causes"[3] was placed on its definition. It may be that the persistence of SIRS over time is a more accurate indication of an inflammatory pathologic process.[12] This is supported by the previous data. The elective group has a high incidence rate of SIRS, but as shown in Fig 3, this is largely during the early postoperative period. In the ruptured group, SIRS persists over a longer time (Fig 4). This would also help to explain the difference in the proportions of each group with SIRS compared with the proportion with multiorgan failure. Because SIRS in the ruptured group is more persistent and more likely to progress to multiorgan failure, it may more accurately reflect inflammation, whereas in the elective group many of the patients with SIRS are probably showing a normal physiologic reaction to surgery.

Table V. Incidence rate of organ failure after AAA repair in three groups of patients

	Elective (*n* = 62)	*Urgent* (*n* = 13)	*Ruptured* (*n* = 25)
Cardiovascular	24 (38.7%)	6 (46.2%)	20 (80%)
Respiratory	8 (12.9%)	5 (38.5%)	21 (84%)
Renal	5 (8.1%)	3 (23.1%)	16 (64%)
Neurologic	0 (0)	1 (7.7%)	1 (4%)
Hematologic	0 (0)	0 (0)	1 (4%)

The assessment of SIRS in this study was performed with only those clinical and laboratory data that would have been routinely collected in these patients if they were not included in this study. This was an attempt to examine how SIRS would perform in a "normal" clinical setting, where frequently some of the criteria that define SIRS may not be available. In this study, these missing data were assumed to be within normal limits. Although this may not have been true in all circumstances, this is the situation that occurs in clinical practice, and because the aim of this study was to determine how SIRS could be applied to routine clinical practice, it was believed that this was the most appropriate solution.

Some patients may have been defined as having organ failure when this was the result of normal physiologic responses to surgery. Cardiovascular failure was responsible for a greater proportion of the total organ failures seen in the elective group than in the urgent and ruptured groups (Table III). The criteria for definition of cardiovascular failure include normocapnic acidosis, severe bradycardia, and hypotension, all of which may transiently occur as part of the physiologic response to AAA repair and not be directly the result of cardiac dysfunction per se. Possible strategies to prevent the false diagnosis of this and other organ failures would have been to use one of the other published systems for defining organ failure, for example, those described by Goris et al[13], Tran et al,[14] or Meesters et al.[15] The system defined by Knaus et al[6] has considerable advantages over the other systems. It is based on a sample of more than 5600 patients; the next largest sample is that of Tran et al,[14] with 497 patients. The system of Knaus et al[6] is also specifically designed to be used in critically ill patients and applied to each 24-hour period of admission to critical care independently. It also assumes that therapies directed towards correction of physiologic derangement are in progress at the time of assessment whereas all of the other systems use these therapeutic interventions as part of their criteria for defining organ failure. All of the systems, other than that of Knaus et al,[6] also use subjective criteria in some of their definitions or physiologic measurements that are not routinely recorded or measured in the critical care units

where the project took place. Each of these systems has merits and disadvantages compared with the system used in this study, and although there may be inaccuracies in absolute numbers seen, the same system has been used for all patients, and therefore, the trends shown are likely to be representative.

The presence of SIRS in a patient after AAA repair is a useful clinical indicator of potential later multiorgan failure, particularly after ruptured AAA repair; however, it is limited by a lack of specificity. It may be possible to increase the specificity of SIRS with analysis of the total number of positive SIRS criteria as a predictive test for organ failure (generating a total SIRS score from 0 to 4). Alternative methods to SIRS, such as APACHE II[16] or SAPS II[17] scoring, may be better predictors of organ failure, but these lack the simplicity of SIRS.

Where use of SIRS as a dichotomous measure does appear to be of use is in identification of those patients who are at very low risk of later organ failure and death by its resolution or absence. The relative simplicity in determination of whether a patient has SIRS or not is one of its key features; it adds very little time to the daily review of a patient on ward rounds. Although many would argue that the SIRS criteria are usually assessed, albeit subconsciously, by any clinician reviewing a patient and that further classification of routine observations is unnecessarily complicating clinical method, we believe that SIRS is a useful method of identifying patients in whom the significance of multiple minor physiologic derangements may be underestimated.

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REFERENCES

1. Sayers RD, Thompson MM, Nasim A, Healey P, Taub N, Bell PRF. Surgical management of 671 abdominal aortic aneurysms: a 13-year review from a single centre. Eur J Vasc Endovasc Surg 1997;13:322-7.
2. McLean RF, Tarshis J, Mazer CD, Szalai JP. Death in two Canadian intensive care units: institutional difference and changes over time. Crit Care Med 2000;28:100-3.
3. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. Crit Care Med 1992;20:864-74.
4. Rodriguez-Gaspar M, Santolaria F, Jarque-Lopez A, Gonzales-Reimers E, Milena A, de la Vega MJ, et al. Prognostic value of cytokines in SIRS general medical patients. Cytokine 2001;15:232-6.
5. Qureshi SS, Lewis SM, Gant VA, Treacher D, Davis BH, Brown KA. Increased distribution and expression of CD64 on blood polymorphonuclear cells from patients with the systemic inflammatory response syndrome (SIRS). Clin Exp Immunol 2001;125:258-65.
6. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. Prognosis in acute organ-system failure. Ann Surg 1985;202:685-93.
7. Pittet D, Rangel-Fausto S, Li N, Tarara D, Costigan M, Rempe L, et al. Systemic inflammatory response syndrome, sepsis, severe sepsis and septic shock: incidence, morbidities and outcomes in surgical ICU patients. Intensive Care Med 1995;21:302-9.
8. Lindsay TF, Luo XP, Lehotay DC, Rubin BB, Anderson M, Walker PM, et al. Ruptured abdominal aortic aneurysm, a "two-hit" ischaemia/reperfusion injury: evidence from an analysis of oxidative products. J Vasc Surg 1999;30:219-28.
9. Moore FA, Moore EE. Evolving concepts in the pathogenesis of postinjury multiple organ failure. Surg Clin North Am 1995;75:257-77.
10. Bone RC. Sir Isaac Newton, sepsis, SIRS and CARS. Crit Care Med 1996;24:1125-8.
11. Godin PJ, Buchman TG. Uncoupling of biological oscillators: a complementary hypothesis concerning the pathogenesis of multiple organ dysfunction syndrome. Crit Care Med 1996;24:1107-16.
12. Talmor M, Hydo L, Barie PS. Relationship of systemic inflammatory response syndrome to organ dysfunction, length of stay, and mortality in critical surgical illness: effect of intensive care unit resuscitation. Arch Surg 1999;134:81-7.
13. Goris RJ, te Boekhorst TP, Nuytinck JK, Gimbere JS. Multiple organ failure. Generalized autodestructive inflammation? Arch Surg 1985; 120:1109-15.
14. Tran DD, Groeneveld AB, van der Meulen J, Nauta JJ, Strack van Schijndel RJ, Thijs LG. Age, chronic disease, sepsis, organ system failure, and mortality in a medical intensive care unit. Crit Care Med 1990;18:474-9.
15. Meesters RC, van der Graaf Y, Vos A, Eikelboom BC. Ruptured aortic aneurysm: early postoperative prediction of mortality using an organ system failure score. Br J Surg 1994;81:512-6.
16. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985;13:818-28.
17. Le Gall JR, Lemeshow S, Saulnier F. A new Simplified Acute Physiology Score (SAPS II) based on a European/North American Multicenter Study. JAMA 1993;270:2957-63.

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